Foreign Animal Disease Report

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In This Issue

Emergency Field Investigations

During the period from October 1, 1987, through September 30, 1988, 260 emergency investigations of suspected exotic diseases were conducted in the United States and Puerto Rico. Of the total, 157 investigations were for suspected vesicular diseases, 38 for septicemic conditions in swine, 3 for mucosal diseases, 50 for Newcastle disease and other poultry diseases, and 12 for suspected screwworm myiasis and other conditions.

A total of 6 cases of exotic Newcastle disease was confirmed in young pet birds: 2 in California, and 1 in Illinois, Michigan, Nevada, and Texas. Available information indicates that these birds may have entered the United States without meeting import health requirements. The infected birds were destroyed. Intensive surveillance and epidemiology indicated there was no spread to other birds.

Seven investigations were conducted for suspected avian influenza (AI). There were no cases of highly pathogenic AI in chickens. However, AI virus was isolated from chickens from two premises in Florida. The viruses isolated were not pathogenic to susceptible chickens in the laboratory, nor were symptoms or lesions observed in the chickens from which the isolates were made.
(Dr. M. A. Mixson, (301) 436-8073)

Vesicular Stomatitis on Ossabaw Island

In July 1988, New Jersey vesicular stomatitis virus (VSV) was isolated from a sand fly and from swine on Osabaw Island, a small island located off the coast of Georgia.

Ossabaw Island has been studied more than any enzootic focus of VSV in the United States. Serological data shows occurrence of the virus annually since monitoring began in 1980. VSV has been isolated from animals on the island on 5 occasions during 3 years (1983, 1987, and 1988). Although cattle, horses, donkeys, white-tailed deer, raccoons, and wild swine living there have had antibodies to VSV, swine have been the only animals in which lesions have been observed, and these have been few in number. Vesicles have been found only on the snout.

On June 20, 1988, 20 female *Lutzomyia shannoi* sand flies were taken from a trap on Ossabaw Island. VSV was subsequently isolated from a pool (pulverized suspension) of the flies at the National Veterinary Services Laboratory (NVSL), Ames, Iowa. The site where the flies were collected was seven-tenths of a mile from the location where clinical vesicular stomatitis was first observed in three wild swine on June 26, 1988. A second

isolation was made from affected swine on July 7, 1988. By July 22, 1988, nine swine had been observed with lesions. Subsequently, VSV was isolated from a second pool of sand flies. All isolates have been New Jersey-type VSV.

Insect trapping was initiated on Ossabaw Island on April 6, 1988, and, contrary to previous years, *Lutzomyia* were immediately found. Their first appearances in 1987 were on April 11, 14, and 30. This year, trapped flies were frozen in the field and kept frozen during sorting. It is suspected that virus was being lost in handling in earlier years due to freezing, thawing, sorting, and refreezing. By July 22, 1988, 221 pools of *Lutzomyia* had been submitted to NVSL for virus isolation.

As in past years, both wild and domestic sentinel swine were used to monitor for seroconversions. This year, domestic sentinels are being kept in pens on the ground and will be used to determine site specificity of VSV activity. In the past, domestic swine were kept in elevated pens so that their only potential exposure to VSV was through flying insects. Serologic data for 1988 was not available at the time this article was written.

A colony of *Lutzomyia* from Ossabaw Island has been started in the insectary at the University of Georgia College of Veterinary Medicine. Study of possible transovarial transmission of the Ossabaw Island strain of VSV is planned. An updated report, which will include an evaluation of this year's serologic and virus isolation results, will be submitted after the laboratory tests have been completed.
(Dr. Victor Nettles, Director, Southeastern Cooperative Wildlife Disease Study, Athens, GA 30602)

(Vesicular stomatitis has not been identified in the continental United States since May 1986. Editor)

Avian Salmonellosis

Salmonellosis caused by phage type 4 *Salmonella enteritidis* has not been detected in the United States, although it has become a major infection in chickens in some areas of Europe, including the Balkan countries, the Iberian Peninsula, and the United Kingdom. It has become the major salmonellosis in humans in England and Wales.

Clinical signs are largely confined to young birds. The disease may cause mortality ranging to 20 percent of an affected flock in the first 1 to 4 weeks of life. Marked stunting may occur in 5 percent or more of infected flocks, but mortality may occur without obvious premonitory signs. Clinical disease is not common in affected semimature or mature chickens. The bacterium has been isolated from the ovaries of hens.

Prominent signs in chicks include pericarditis, perihepatitis, airsacculitis, and retained inspissated yolk sacs. This range of lesions resembles the lesions of colibacillosis. Pericarditis in survivors may persist up to the time of slaughter.

Horizontal transmission occurs readily within an affected flock, but transmission from flock to flock is uncertain. Vertical transmission appears to be the major means of dissemination, probably by both true transovarial transmission and by egg-shell penetration. As with other paratyphoid salmonelloses, infection can be expected to persist in a flock throughout life.

The characteristics of infection in other avian species and in domestic and wild mammals have not been established. One might expect that a range of birds and mammals would be susceptible to infection with or without clinical disease.

The bacterium is present in large numbers in lesions and can be isolated from those sites, usually in pure culture. Several swab samples from lesions may be placed in transport medium for later isolation attempts, or intact birds can be submitted directly to a diagnostic laboratory. Infected adult chickens may develop antibodies that cross-react with *S. pullorum* antigens, but diagnostic dependability has not been established. Isolation and phage typing is necessary to establish a diagnosis of salmonellosis caused by phage type 4 *Salmonella enteritidis*.
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Foreign Animal Disease Update

In South America, during April, May, and June 1988, Brazil reported 96 herds affected with vesicular disease(**foot-and-mouth disease** (**FMD**) types O_1 and C_3), Argentina reported 82 herds affected, Bolivia reported 22 herds (FMD type A_{24} and C_3), Ecuador reported 22 herds (FMD type O), Paraguay reported 1 herd (FMD type not specified), and Colombia reported 177 herds (FMD types O_1, and A_{24}, and **vesicular stomatitis**). In Colombia, 11 herds were affected with New Jersey strain VS and 36 with Indiana strain. According to the Office International des Epizooties (OIE), Chile is considered free of FMD. However, the United States does not recognize that country as free of FMD. The last slaughtering of diseased animals there was on August 20, 1987. On September 30, 1987, a group of Chilean animals that had reacted to the VIA (virus infection-associated antigen) test for FMD was also slaughtered.

Italy reported a second outbreak of FMD type C_1 July 11, 1988 (see 16-3:3). The outbreak was located 5 km from the site of an outbreak on June 24, 1988, in the Tuscany Region. To stamp out the disease, 34 cattle and 1,500 swine were destroyed.

According to OIE, the Federal Republic of Germany is again free of FMD, as 6 months have elapsed since the last case was eliminated. No outbreaks have been reported there since January 12, 1988. However, the United States does not recognize that country as free of FMD.

Israel reported three outbreaks of FMD type O, Manissa strain, during June and July 1988, in the northern districts of Tsefat and Golan. Type O FMD virus was also isolated in June from Jordanian samples sent to the World Reference Laboratory, Pirbright, England.

Kuwait continues to report outbreaks of FMD virus type O. By June 20, 1988, 27 farms had been affected, involving cattle, swine, sheep, and goats. Samples submitted to Pirbright for subtyping were found to be antigenically related to O_1 BFS and O_1 Manissa. Susceptible livestock were being vaccinated with O_1 Manissa monovalent vaccine in an effort to halt spread of the disease.

The World Reference Laboratory also reported the following for the months of April, May, and June 1988: Type O - Bahrain, Turkey, Pakistan, Niger, and Hong Kong; Type A - Turkey; Type C - Philippines; and, Type SAT_1 - Zambia.

Sri Lanka, an island country off the southern tip of India, reported cases of **rinderpest** (**RP**) during the first 4 months of 1988. The first cases of RP were reported in Sri Lanka in December 1987. Uganda, in Eastern Africa, reported outbreaks of RP in April and May 1988.

Senegal, in Western Africa, and Oman, in the Arabian Peninsula, reported outbreaks of **peste des petits ruminants** during March and April, respectively.

During the first 6 months of 1988, **contagious bovine pleuropneumonia** was reported in the African countries of Burkina Faso, Angola, Mali, and Namibia; and in Kuwait, on the Arabian Peninsula.

Lumpy skin disease was reported in Kuwait, Senegal, Mali, Angola, Namibia, Zaire, Republic of South Africa, and Madagascar during the first 6 months of 1988.

Also, during the first 6 months of 1988, outbreaks of **sheep and goat pox** were reported in Greece, Turkey, Morocco, Senegal, Mali, Tunisia, Kuwait, Oman, and Pakistan.

African horse sickness (**AHS**) was reported in Namibia and Republic of South Africa during the first 6 months of 1988. AHS was reported in Spain during the fall of 1988.

In June 1988, Italy reported an outbreak of **African swine fever** (**ASF**) in the Sardinia Region. As a result of the outbreak, 83 pigs were slaughtered and buried. In April and May 1988, Portugal reported 28 outbreaks of ASF. A total of 1,463 swine was destroyed in Portugal during these 2 months. In March and April 1988, Spain reported 14,264 swine destroyed in 76 outbreaks of ASF. Angola and South Africa also reported ASF during the first 6 months of 1988.

Hog cholera was reported by the following countries during the first 6 months of 1988: Federal Republic of Germany, Italy, Czechoslovakia, Yugoslavia, Mexico, Colombia, Ecuador, Peru, Chile, Brazil, Argentina, Uruguay, Paraguay, Sri Lanka, Taiwan, Korea, Malaysia, and Madagascar.

Teschen disease was reported in Madagascar in January 1988, and in the Ukraine, USSR, in March 1988.

South Africa, Namibia, and Senegal reported cases of **Rift Valley fever** March through June 1988.

Switzerland reported its first cases of **contagious equine metritis** (**CEM**) on April 11, 1988. The outbreak affected five cross-bred stallions on two stud farms, as well as the mares covered by these stallions since March 1, 1988. To prevent further spread of the disease, the 120 horses involved in the outbreak have been excluded from breeding.

The Netherlands reported an outbreak of CEM during June 1988. The disease first appeared there in July 1987. An infected stallion covered 42 mares in the Netherlands and 53 in Belgium, and they are suspected to be infected with the causal bacteria. To prevent further spread of the disease, these mares will be bred only by artificial insemination. As of July 27, 1988, 12 of the mares in the Netherlands had been shown to have CEM.
(Dr. Percy W. Hawkes, (301) 436-8285)

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Bovine Spongiform Encephalopathy

Bovine spongiform encephalopathy (BSE) of domestic cattle was first diagnosed in November 1986, by workers at the Central Veterinary Laboratory, Weybridge, England. The disease is characterized by grey matter spongiosis and neuronal vacuolation. The associated neurological syndrome that was observed by Veterinary Investigation Officers had an insidious onset, slow progress, and fatal outcome. A preliminary report of initial cases of the disorder suggested that it had a close similarity to

scrapie of sheep and proposed the name bovine spongiform encephalopathy (*Wells, G. A. H.; Scott, A. C.;Johnson, C. T.; Gunning, R. F.; Hancock, R. D.; Jeffrey, M.; Dawson, M.; and Bradley, R., 1987. Vet. Rec., 121: 419-420).*

Sporadic cases continued to be reported in 1987, and by June of that year, an epidemiological investigation was initiated. By the end of the year, over 100 cases had been confirmed from most parts of Great Britain, although the incidence remained greatest in the southern counties of England. By mid-1988, the incidence of BSE had risen to 500 confirmed cases among a total adult cattle population of 4 million. In June 1988, BSE was legislated a notifiable disease. There have been no reports of a similar natural disorder of cattle elsewhere in the world.

BSE Epidemiology

Epidemiological studies (*Wilesmith, J.; Wells, G. A. H.; Cranwell, M. D.; and Ryan, J. B. M. Vet. Rec., in press*) suggest that BSE is new and that the first clinically suspected case occurred in April 1985. While some domestic cattle herds have experienced multiple cases of BSE, incidence within herds is generally low. Single cases were recorded in 75 percent of the affected herds. Within-herd incidence is significantly higher in dairy herds than in beef cow-calf herds. The disease was confined to adult cattle, and no associations with breed, sex, or stage of lactation or pregnancy were observed. Epidemiologically, BSE is an extended common-source epidemic with no evidence of cattle-to-cattle transmission, so that each affected animal represents an index case. The inquiry eliminated a number of epidemiological factors, namely introduction of disease by imported animals or semen, common management practices, and exclusive causation by simple autosomal modes of inheritance.

A computer-based simulation model of the data suggests that BSE is a new disease of cattle of food-borne origin and that British cattle were first exposed in 1981 or 1982. Analysis of age-specific incidences indicates an incubation period from 2 to 8 years and a risk of exposure for calves of 30 times that for adults.

Given the nature of the disorder, it seems probable that such exposure may emanate from scrapie-infected material in the diet. There is evidence of a food-borne source, and brain extracts from affected cows have fibrils structurally and chemically common with Scrapie Associated Fibrils (SAF) of scrapie. *(Wells, G. A. H.; Scott, A. C.; Johnson, C. T.; Gunning, R. F.; Hancock, R. D.; Jeffrey, M.; Dawson, M.; and Bradley, R., 1987. Vet Rec., 121: 419-420. Wells, G. A. H.; and Scott, A. C., 1988. Neuropath. Appl. Neurobiol., 14: 247. Hope, J.; Reekie, L. J. D.; Hunter, N.; Multhaup, G., Beyreuther, K.; White, H.; Scott, A. C.; Stack, M. J.; Dawson, M.; and Wells, G. A. H., Nature, in press).*

The recent preliminary report of production of scrapie-like disease in mice inoculated with brain homogenates from BSE cases provides the first direct evidence that BSE is a transmissible disease (*Fraser, H.; McConnel, I.; Wells, G. A. H.; and Dawson, M. 1988 Vet. Rec., 123: 472).*

British Regulatory Actions

Because animal proteins were judged the most likely vehicle for the agent of BSE, withdrawal of animal proteins from ruminant rations was temporarily imposed by the British Government. Further investigations are in progress to more adequately identify the disease by its characteristics, its cause, and its transmission. The British Government also ordered destruction of affected cattle to prevent contamination of the human food chain and to avoid recycling of ruminant animal protein.

The BSE epidemic is being monitored by required reporting of clinically suspect cases. Monitoring the disease status of the progeny of affected cows is of vital interest to deter-

mine possible occurrence of maternal BSE transmission and to devise control strategies.

Clinical Signs

The clinical signs of BSE are mainly neurological. Changes are noted in behavior, posture, movement, and sensory perception, all of which indicate a diffuse disorder of the central nervous system. The earliest signs are commonly behavioral, resembling those of hypomagnesemia or nervous ketosis, but may also include persistent kicking during milking, gait ataxia, reduced milk yield, and loss of body weight. Apprehensive behavior, gait ataxia, and loss of general bodily condition are the most frequently reported signs throughout the course of the disease. Apprehension may progress to displays of overt frenzy.

There are certain similarities between clinical features of BSE and rabies, especially the furious form of rabies, and, furthermore, only a very small proportion of BSE cases die early in the disease course. Together, these findings presage difficulty in clinically differentiating these two diseases, if BSE were to occur where rabies is endemic.

Other frequent signs are exaggerated ear movements and aggression to or avoidance of other cows.

Abnormal gait accompanies the onset of behavioral changes and sometimes becomes the dominant sign later in the course of the disease. Ataxia, usually mild at first and confined to the pelvic limbs, is evident as swaying of the hind quarters and exaggerated forward motions of the lower limbs. Similar motions of the forelimbs may follow, with occasional falling of some affected cattle as the disease progresses. Later, generalized paresis results in more frequent falling and, ultimately, inability to stand. Tremors and twitching of muscle groups of mainly the head and shoulders are also seen. Increased sensitivity to stimulation by touch and sound is evident in most cases. Handling of the head is vigorously resisted, and handling of the udder at milking time may evoke violent kicking. Excessive grooming, rubbing, or scratching activity occurs in some instances, but pruritus, as observed in scrapie of sheep, is not a prominent feature. Sudden loud noise may in some cases induce immediate falling. As the disease progresses, animals that are unmanageable, paretic, or cachetic are ordinarily humanely destroyed.

Location and Cause

There have been no reports of a similar natural disease of cattle in the United States. However, the notion that such a disorder may exist has been raised by a report that transmissible spongiform encephalopathy in ranch-reared mink (TME) may be attributable to the feeding of cattle carcasses, and not to the feeding of scrapie-contaminated sheep or goat tissues previously considered the most probable source of TME. (Marsh, R. F.; and Hartsough, G. R., 1986. Proceedings of the Seventh Annual Western Conference for Food Animal Veterinary Medicine, page 20, University of Arizona, Tucson. Marsh, R. F, and Hartsough, G. R., 1988. Proceedings of the Fourth International Scientific Congress in Fur Animal Production, pages 204-207. Canadian Mink Breeders Association, Toronto.) Prior to these reports, the feeding of scrapie-contaminated sheep or goat tissues was considered the probable cause of TME.
(*By personal communication, Dr. R. F. Marsh reports that the possibility that the mink were also fed bone meal and other meat byproducts other than bovine byproducts was not excluded. It would be premature to conclude that TME was transmitted from cattle. Editor*)

Pathological and epidemiological observations suggest that BSE is a new member of

that group of disease caused by unconventional viral agents or "prions" which comprises scrapie of sheep and goats, chronic wasting disease of mule deer, TME of ranch-reared mink, and Kuru and Creutzfeldt- Jakob disease of humans. A substantial research program on BSE, including transmissibility studies, has been established in England.
(G. A. H. Wells, Central Veterinary Laboratory, New Haw, Weybridge, Surrey KT153NB, England)

Focus on African Horse Sickness

African horse sickness (AHS) is a disease of equines caused by an arthropod-borne virus. This virus is classified as an orbivirus in the Reoviridae family with the viruses of bluetongue, Ibaraki disease, epizootic hemorrhagic disease of deer, and a number of other related viruses. In horses, the disease is usually characterized by fever, edema of subcutaneous tissues and lungs, hemorrhages of the heart and digestive system, and high mortality. In mules and donkeys, the disease is less severe than in horses; in zebras, it may occur as an inapparent infection.

History

A devastating new disease of horses reported in the records of the Cape Colony of South Africa in 1719 was later considered to be the first report of AHS. About 1,700 horses imported by the Dutch East Indies Company died. Thereafter, the disease recurred annually. It appeared after the spring rains and continued until the first frost.

Epizootics with substantial losses occurred at about 20-year intervals in the Cape Colony. The epizootic of 1854-55 was particularly damaging. Nearly 70,000, or more than 40 percent of the total horse population of the Cape of Good Hope, died of the disease.

AHS was soon found to be enzootic in most of Africa south of the Sahara; it has had a profound impact on the history of the continent. Because mortality among horses was close to 90 percent, early explorers rode oxen, navigated rivers, or walked; military expeditions were conducted without mounted cavalry, and early settlers were often obliged to use animals other than horses to till their fields.

AHS has occasionally spread beyond its usual confines. In 1928, it spread up the Nile valley from Sudan to Egypt. The number of animals involved was not large, but 89 percent of the horses and 70 percent of the mules that contracted the disease died. Some donkeys with AHS also died. No new cases occurred after the onset of winter, and the region remained free of this disease during the next 15 years. AHS reappeared in Sudan in the summer of 1943, and spread north to the Nile delta. It disappeared during the winter, recurred in 1944, and spread to Palestine, Syria, and Jordan. Although losses were not large, these occurrences demonstrate that AHS is capable of spreading rapidly and extensively.

A major epizootic occurred in 1959-60, first in Iran, and then in West Pakistan and Afghanistan during the summer of 1959. In the spring of 1960, the disease spread rapidly to India, Turkey, Cyprus, Iraq, Syria, Lebanon, and Jordan. An estimated total 300,000 equines died of the disease.

The last widespread epizootic occurred in 1966-1967. Algeria, Morocco, Tunisia, and Spain were affected. However, vaccines were available by that time, and control methods were fairly well established. Even though losses were undoubtedly reduced, the epizootic was very costly.

The most recent excursion of AHS outside the continent of Africa began in Spain during the summer of 1987. At that time (*See 15-4:4*), 300 horses either died or were destroyed due to AHS. The virus apparently came from Africa in zebras imported from the Republic of South Africa. The zebras were brought via Portugal to Spain, where they were placed in zoos and a wild animal park. Equines in the area of the outbreak were vaccinated. (*See article on page 4 in this issue: Foreign Animal Disease Update.*)

AHS Virus

The viruses of AHS (AHSV) and bluetongue virus (BTV) are almost indistinguishable by physical-chemical means. Both AHSV and BTV are orbiviruses, and consist of 10 segments of double-stranded RNA surrounded by a double-layered protein shell. They are 70-80 mm in diameter, have large doughnut-shaped capsomeres, and are ether-resistant and acid-labile. Some hybridization between the nucleic acids of AHSV and BTV has been demonstrated. Both are transmitted mainly by biting midges (*Culicoides*). However, the host ranges of AHS and bluetongue are different, and they are serologically distinct.

Isolates of AHSV can be identified by the complement fixation (CF) test. However, marked differences in the efficacy of the vaccines that were produced from different isolates indicated that there might be several serological types of the virus. So far, nine serological types have been identified by neutralization tests.

AHSV Hosts

Natural infections with AHSV have been found in equines, dogs, and camels. Among the equines, susceptibility is highest in horses, somewhat lower in mules, and lowest in donkeys. Mortality rates are ranked in the same order, with rates as high as 95 percent for horses and about 80 percent for mules. African donkeys are quite resistant to AHS, and few deaths have occurred among them. Mortality rates have varied considerably from one epizootic to another, but they have always followed the same general pattern of host susceptibility. However, in the 1960 epizootic in the Middle East, substantial losses occurred among donkeys. Zebras are normally highly resistant, but some deaths from AHS have been reported.

The susceptiblity of dogs to AHS was observed over the years by a number of investigators. They generally believed that the disease could be acquired by dogs only by eating infected meat or by being experimentally injected with AHSV. In a search for animals other than equines that might harbor the virus over the winter, Egyptian workers examined 111 blood samples collected during the winter from street dogs in an enzootic area in Aswan Province. Isolations of AHSV were made from three of the samples, thus proving that some dogs, at least in that area, were naturally infected and carried the virus during the winter. Two AHSV isolations were also made from camels in the Aswan Province of Egypt.

In the search for reservoir hosts in Africa, sera of wild and domesticated animals were tested in limited surveys for the presence of AHSV-specific antibodies. Significant antibody levels were found in elephants and zebra. The virus was isolated from zebra but not from elephants. In Egypt, antibodies to AHS were found in sheep (23.5 percent of total animals tested), goats (14 percent), dogs(7 percent), camels (5 percent), and buffalo (4 percent). Antibodies were not found in cattle. Judging from the relatively high levels of antibody found in sheep and goats, they too may be naturally infected.

A wide variety of animals have been experimentally infected with AHSV. White mice were found to be susceptible to intracerebral inoculation of AHSV. Serial passage by that route produced neurotropic AHSV. With the exception of rabbits, most common laboratory animals (guinea pigs, hamsters, and rats) may be infected with mouse-adapted

neurotropic strains of AHSV. Infections with AHSV have not been reported in humans.

AHSV Transmission

Because of its seasonal occurrence and the fact that it did not spread by contact, AHSV was assumed to be transmitted by biting insects. The successful protection of horses against the disease by confining them in mosquito-proof stables during the night led to a suspicion that the vector was a nocturnal bloodsucking arthropod. Although a wide variety of arthropods have been suggested as possible vectors of AHS, species of Culicoides are generally accepted as the natural vector. This conclusion is based on studies conducted in South Africa, where wild-caught Culicoides were allowed to feed on a susceptible horse that died of AHS 12 days later.

All major and sporadic outbreaks in Egypt have begun in the Aswan and Qena Provinces and in the border lands between Egypt and Sudan. Some sporadic cases may be attributed to illegal entry of infected equines from Sudan and failure to vaccinate all susceptible equines. The disease periodically recurs during hot weather following heavy rains. Mosquitoes and Culicoides abound under these conditions, but they have not been shown to harbor AHSV during cool, dry seasons.

AHSV Reservoir

Egyptian workers have tried to identify an animal reservoir for AHSV during the off-seasons. Their attention focused especially on dogs and camels. Dogs were known to be susceptible to AHS and were numerous in the area. Camels were often brought in from Sudan, where AHS control measures were far less stringent. The 23 percent incidence of AHSV-specific CF antibodies in Sudanese camel blood was significantly higher than that found in Egyptian camels (5 percent). The search yielded six AHSV isolations from dogs and four from camels. The camel isolations were particularly significant because two were obtained from engorged *Hyalomma dromedarii* ticks removed from camels imported from Sudan. The infected camels appeared normal. *Hyalomma dromedarii* larvae and nymphs that were fed on infected animals later transmitted the disease to susceptible animals. Nymphs continued to transmit the disease when they became adults.

AHS Pathogenesis

Four forms of AHS have been distinguished in horses:

<u>The peracute or pulmonary form</u>. This form of AHS is the most common of the four. It is usually seen in severe epizootics where mortality rates are high. An incubation period lasting 3 to 5 days precedes an acute febrile reaction that may last only 1 or 2 days, with temperatures as high as 40° to 40.5°C (104° to 105°F). This is followed by progressive respiratory disease, usually including severe dyspnea and spasmotic coughing. The animal stands with legs spread apart, head extended, and nostrils dilated.

Moribund animals are unable to stand, and, at the time of death, a frothy liquid may flow from their mouths and they drown in their own fluids. The mortality rate is over 90 percent.

The most characteristic changes seen at necropsy are edema of the lungs and serous fluid in the pleural cavity. The lymph nodes, especially those in the thoracic and abdominal cavities, are enlarged and edematous. Periaortic and peritracheal edematous infiltration, hyperemia of the glandular fundus of the stomach, congestion of the renal cortex, hyperemia and petechial hemorrhages in the mucosa and serosa of the large and small intestines, and subcapsular hemorrhages in the spleen are also commonly seen. Petechial hemorrhages occur in the pericardium, and the pericardial sac may contain fluid. Epicardial and endocardial petechial hemorrhages are occasionally seen, but cardiac lesions are usually not outstanding.

Cardiac or subacute edematous form. Caused by AHSV strains of lower virulence, this form of AHS may occur in immune animals infected with heterologous strains of the virus. The incubation period is about 7 to 14 days, and the first clinical sign is a febrile reaction that lasts from 3 to 6 days.

As the fever begins to subside, characteristic edematous swellings develop. These first appear in the supraorbital fossae and eyelids and then extend to the lips, cheeks, and tongue. Subcutaneous edema may extend down the neck and involve the shoulders, brisket, and thorax. In animals that recover, the edema subsides within 3 to 8 days. The mortality rate is about 50 percent. Death usually occurs within 4 to 8 days after the onset of fever. Before death, petechial hemorrhages appear in the conjunctivae and in the ventral surface of the tongue. Colic may precede death from cardiac failure.

Hydropericardium is the most prominent and constant change seen at necropsy. The pericardial sac may contain more than 2 liters of fluid. Petechiae and ecchymoses are usually present on the epicardium and endocardium. These hemorrhages are often most prominent along the course of the coronary vessels and beneath the bicuspid and tricuspid valves. The lungs may be normal or only slightly congested. There rarely is an excess of fluid in the thoracic activity. The gastrointestinal tract usually has lesions similar to those seen in the pulmonary form of AHS. Submucosal edema is usually much greater in the cardiac form of AHS than in the pulmonary form.

Mixed form. The mixed form of AHS is a combination of the cardiac and pulmonary forms. The majority of fatal cases of AHS may be classified as the mixed form, with lesions of either the pulmonary or cardiac form predominating.

African horse sickness fever. In its mildest form, AHS may appear as no more than a thermal response of 1 to 5 days duration. The temperature may go as high as 40.5°C (105°F), but usually after 2 days, the fever subsides, and the animal recovers. This is the usual form of AHS in experimentally infected goats or donkeys. Naturally occurring AHS fever may escape detection.

AHS in Wildlife

AHSV was clearly present in South Africa before horses were introduced. This conclusion is based upon the absence of any prior report of the disease outside the continent of Africa, and the fact that AHS appeared only after horses were taken into certain areas of that continent. Therefore, a reservoir for the disease agent was sought among indigenous animals. A wildlife reservoir for AHSV has not been definitely established. The zebra was a prime suspect, and some observers reported deaths that they attributed to AHS. Clinically recognizable AHS has been seen in zebra, and AHSV has been isolated from zebra. However, horses also contracted AHS in areas where zebra and other game animals did not exist. In these areas, injection of blood from locally caught small mammals, birds, reptiles, and amphibians into susceptible horses also failed to produce the disease.

Much later, AHSV neutralizing and CF antibodies were found to be quite common in the blood of zebra and elephants in Kenya and South Africa. The presence of antibodies in the elephant has not been explained. AHS has never been observed in elephants, nor has AHSV been isolated from them.

AHS Immunity

Animals that have survived AHS have a solid immunity to the particular virus type involved, but remain susceptible to the other serological types. Foals from immune dams acquire a natural immunity that protects them from the disease for approximately

8 months after birth.

In some enzootic areas, mass vaccination of equines is practiced. Vaccination is seldom done in other animals such as camels, goats, sheep, and dogs that may be infected but seldom display clinical signs.

AHS Diagnosis

In enzootic areas where veterinarians and horse owners are familiar with the disease, clinical signs and gross lesions are usually characteristic enough to permit a presumptive diagnosis. For example, edema of the supraorbital fossae is pathognomonic for the cardiac form of AHS.

Some of the clinical signs and post-mortem findings of the disease may be confused with other equine diseases such as equine infectious anemia, equine piroplasmosis, purpura hemorrhagica, and rhinopneumonitis. Therefore, a definitive diagnosis of AHS requires isolation and identification of the virus.

AHSV isolation is usually achieved by intracerebral inoculation of unweaned mice with defibrinated blood taken at the peak of fever. Spleen suspensions have also been used for viral isolation. A litter of 8 to 10 mice is used for this purpose. Each mouse is given 0.025 ml of blood, diluted in 10 parts sterile distilled water or phosphate buffered saline. The mice are observed for 2 weeks, and the brains of those that show nervous signs and prostration are removed and inoculated intracerebally as a 10 percent suspension into another litter of suckling mice. After 3 to 5 serial passages, mouse mortality is usually 100 percent. Viruses isolated in this manner remain antigenic, even though they are neurotropic and may no longer produce clinical disease in horses.

Antigens for the CF test, Outcherlony agar gel immunodiffusion test (AGID), and virus neutralization test (VN) have been prepared from laboratory-infected mouse brains. Stocks of reference viruses, also prepared in mice, have been used to produce type-specific antisera in rabbits. These procedures, reagents, and tests can be used to isolate, identify, and type AHS viruses. They also can be used to survey animal populations for AHS antibodies. The CF test is useful for rapid diagnosis. It is group specific, but limited by the short period during which CF antibodies are present in the serum of infected animals. For typing, the VN test must be used. Virus-neutralizing antibodies are present for a much longer time than CF antibodies.

Cell cultures were eventually used to isolate AHSV directly from naturally infected animals and to type viruses by VN. This improves upon the slow and tedious procedures of propagating AHSV by mouse inoculation. Of the numerous cell cultures tested, stable monkey kidney cell lines, MS and VERO, and baby hamster kidney cell line, BHK21, proved to be most useful.

Coexistence of virus and antibody in the blood of the infected animal accounts for some of the difficulty in isolating the virus. However, most of the virus in the blood appears to be firmly associated with erythrocytes. These may be washed comparatively free of antibody. Virus isolation is facilitated by using an inoculum of washed erythrocytes, hemolyzed either by sonication or addition of distilled water. The use of roller-tube cultures also appears to favor virus isolation.

Fluorescent antibody (FA) techniques also have been applied to AHSV detection. Although the FA techniques do not identify AHSV types, they are more convenient and rapid than CF in identifying AHSV group antigens. Indirect immunofluorescence has been used successfully in a survey for AHS antibodies in wild zebra in Kenya.

A number of new tests are being developed. Promising preliminary trials have been conducted with some of them. Much more testing will be required before they are likely to be accepted as standard procedures. Among them are microadaptations of the CF and AGID tests, hemagglutination tests with erythrocytes coupled to type-specific AHS antibodies for the typing of AHSV isolates, and an indirect enzyme-linked immunosorbent assay (ELISA).

AHS Control

Because AHSV is arthropod-borne, both its vectors and its vertebrate hosts are considered in prevention, control, and eradication. Repellents and insecticides apparently have reduced the incidence of the disease substantially, although controlled studies were not made. Some countries spray the interiors of airliners with an insecticide aerosol before passengers disembark from flights originating in regions where AHS exists.

In the United States, horses from Africa, Asia, and the Mediterranean countries are quarantined on arrival in insect-proof stables. They remain for at least 60 days before being allowed to proceed to their destination.

A number of satisfactory vaccines are now available. However, vaccine supplies may not be sufficient to quickly arrest a fast-spreading major epizootic. When AHS is first diagnosed in an area, affected horses should be quickly and humanely eliminated, and the uninfected equines should be vaccinated with a polyvalent vaccine. After the diagnosis of AHS has been confirmed and the virus type identified, a homologous vaccine may be substituted for the polyvalent vaccine to reduce cost and improve efficiency.

Conclusion

African horse sickness continues to be enzootic in most of Africa, but losses are generally kept at a minimum through the use of vaccines. Although no major epizootics had been reported outside of Africa in 20 years, a 1987 outbreak in Spain served as a reminder that it is still too early to relegate the disease to history.

In most of the world, the horse is no longer used as a beast of burden and is no longer of military importance. However, it has become of great importance for sport and recreation. Valuable horses are often transported by air from one country to another, despite the fact that there is still much to be learned about some of the diseases that may be travelling with them.

The reservoirs and vectors of AHSV are not well established. AHS is a disease that deserves more attention than it may now be receiving, especially from countries outside the African continent.

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Subject Index

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